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1: Oncogene 2002 Oct 7;21(45):6870-6

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Molecular epidemiology of smoking and lung cancer.

Shields PG.

Lombardi Cancer Center, Georgetown University Medical Center, The Research Building, W315, 3970 Reservoir Rd. NW, Washington, DC 20007, USA.

Lung cancer is the single most common cause of death, and almost all of it is due to tobacco smoking. Before the widespread use of cigarettes in this century, lung cancer was a rare illness. Tobacco smoke is a complex mixture of numerous mutagens and carcinogens. Over the last 40 years, the type of cigarettes most frequently used has been changing, namely the increased use of low tar and nicotine cigarettes. This has been accompanied by an increased risk of lung cancer due to a smokers' need to maintain blood nicotine levels, which in turn causes the need for smoking more cigarettes per day and deeper inhalation. This phenomena has led to the increasing rates of lung adenocarcinoma, compared to squamous cell carcinoma. It also probably explains, in part, the greater risk of lung cancer in women compared to men (in addition to some biological differences). The study of lung cancer involves many types of biomarkers, including those that measure exposure, the biologically effective dose and harm. The use of these has allowed us to understand many parts of lung carcinogenesis. Genetic susceptibilities play a large role in lung cancer risk. They govern smoking behavior (affecting dopamine reward mechanisms due to nicotine and nicotine metabolism), carcinogen metabolism and detoxification, DNA repair, cell cycle control and other cellular responses. The need for the study of lung cancer is highlighted by the need to improve cessation rates and reduce exposure among persons who cannot quit smoking, for better prevention strategies for former smokers and an understanding of environmental tobacco smoke risk. doi:10.1038/sj.onc.1205832

PMID: 12362269 [PubMed - in process]

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